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<a href="#">#30 Search Lu HH</a>		12:38:21	104
<a href="#">#27 Search Selby M and HCV</a>		12:35:31	12
<a href="#">#26 Search Selby M</a>		12:35:24	94
<a href="#">#25 Search PKR and HCV subgenomic</a>		12:34:53	6
<a href="#">#23 Search PKR and HCV</a>		12:34:39	81
<a href="#">#24 Search PKR and HCV replicon</a>		12:33:52	9
<a href="#">#21 Search Dubensky 1996 and sindibis virus</a>		12:31:37	2
<a href="#">#19 Search Behrens 1998 and pestivirus</a>		12:30:30	2
<a href="#">#18 Search Behrens 1998</a>		12:30:18	123
<a href="#">#17 Search Behrens 1998 and HCV</a>		12:30:09	0
<a href="#">#14 Search HBsAg and ayw1/ayw2</a>		07:18:48	11
<a href="#">#10 Search HBV and ayw1/ayw2 variant</a>		07:17:58	0
<a href="#">#9 Search HBsAg and ayw1/ayw2 variant</a>		07:17:52	0
<a href="#">#8 Search HBV ayw1/ayw2 variant</a>		07:17:41	0
<a href="#">#3 Search hbv and sudan</a>		07:16:08	6
<a href="#">#2 Search HBV and sudan</a>		07:16:06	0
<a href="#">#1 Search HBV 29681 strain</a>		07:15:57	0

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<a href="#">#10</a>	<a href="#">Search PKR knock out Limits: Publication Date to 2000/08/04</a>	12:50:38	<a href="#">1</a>
<a href="#">#9</a>	<a href="#">Search PKR knock out and virus infection Limits: Publication Date to 2000/08/04</a>	12:50:31	<a href="#">0</a>
<a href="#">#2</a>	<a href="#">Search PKR mutant and virus infection Field: All Fields, Limits: Publication Date to 2000/08/04</a>	12:46:18	<a href="#">9</a>
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(FILE 'HOME' ENTERED AT 13:05:59 ON 05 JAN 2006)

FILE 'CAPLUS' ENTERED AT 13:06:07 ON 05 JAN 2006

L1           210 "HOUSE KEEPING GENE"  
L2           2055 "ANTI-VIRAL"  
L3           1 L1 AND L2  
L4           13 MUTATION AND L1  
L5           51207 LETHAL  
L6           0 L1 AND L5  
L7           285870 MUTATION  
L8           13 L1 AND L7  
L9           2663 IL2  
L10          1 L1 AND L2  
L11          0 INF GENE MUTATION  
L12          51207 LETHAL  
L13          32950 GENE (W) MUTATION  
L14          537 L12 AND L13  
L15          65045 CELL (S) DEATH  
L16          9 L14 AND L15  
L17          2 L1 AND L15  
L18          0 IL2 KNOCK OUT MICE  
L19          2663 IL2  
L20          6972 KNOCK OUT  
L21          2 L19 AND L20  
L22          977 IL8  
L23          0 L22 AND L20  
L24          0 L20 AND L22  
L25          3858 CCR5  
L26          2 L25 AND L20  
L27          53 IL8 RECEPTOR  
L28          0 L27 AND L20  
L29          51 INTERLEUKINE  
L30          0 L29 AND L20  
L31          164691 CYTOKINE  
L32          340 L31 AND L20  
L33          137349 DEATH  
L34          24 L33 AND L32

FILE 'CAPPLUS, BIOSIS' ENTERED AT 11:10:04 ON 05 JAN 2006

L1        144 "ANTI VIRAL RESPONSE"  
L2        11387 KNOCK (W) OUT  
L3        0 L1 AND L2  
L4        7 PKR AND L1  
L5        203666 VIRUS (S) INFECTION  
L6        2 L4 AND L5  
L7        0 HCV AND L4  
L8        297 PKR (S) MUTANT  
L9        90 INFECTION AND L8  
L10      1097807 VIRUS  
L11      82 L9 AND L10  
L12      2 HCV AND L11

FILE 'STNGUIDE' ENTERED AT 11:22:11 ON 05 JAN 2006

L13      0 CELL AND L11  
L14      0 TRANSFECTED

FILE 'CAPPLUS, BIOSIS' ENTERED AT 11:27:02 ON 05 JAN 2006

L15      6770361 CELL  
L16      79 L15 AND L11  
L17      79 INFECTION AND L16  
L18      7412 REPLICON  
L19      0 L18 AND L17  
L20      0 TRANSFECTION AND L19  
L21      1 HCV AND L17  
L22      39 "DOMINANT NEGATIVE PKR"  
L23      12 L17 AND L22

FILE 'STNGUIDE' ENTERED AT 11:31:21 ON 05 JAN 2006

ACCESSION NUMBER: 2002;428614 CAPLUS

DOCUMENT NUMBER: 137:5026

TITLE: NOG immunodeficient mouse for disease models and human antibody production

INVENTOR(S): Ito, Mamoru; Kobayashi, Kimio; Nakahata, Tatsutoshi;

Tsugi, Koichiro; Habu, Sonoko; Koyanagi, Yoshio;

Yamamoto, Naoki; Sugamura, Kazuo; Ando, Kiyoshi

PATENT ASSIGNEE(S): Central Institute for Experimental Animals, Japan

SOURCE: PCT Int. Appl., 86 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: Japanese

FAMILY ACC. NUM. COUNT: 1

## PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002043477	A1	20020606	WO 2001-JP9401	20011025
W: CA, JP, US RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR				
CA 2402459	AA	20020606	CA 2001-2402459	20011025
EP 1338198	A1	20030827	EP 2001-978918	20011025
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
US 2003182671	A1	20030925	US 2002-221549	20020910
PRIORITY APPLN. INFO.:			JP 2000-367296	A 20001201
			WO 2001-JP9401	W 20011025

AB An immunodeficient mouse is established by cross intercross of NOG/Shi mouse, SCID mouse, and IL-2R $\gamma$  knock-out mouse (NOG mouse). The NOG mouse shows functional deficiency of T and B lymphocytes, NK cells, macrophages, and dendritic cells. Transplantation of human stem cells to NOG mouse shows engagement of the cells without rejection. It is useful in constructing human antibody, stem cell assay system, disease models and drug screening for leukemia and AIDS.

REFERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L34 ANSWER 17 OF 24 CAPLUS COPYRIGHT 2006 ACS on STN  
ACCESSION NUMBER: 2002:411839 CAPLUS  
DOCUMENT NUMBER: 137:76241  
TITLE: Caspases - their role in apoptosis and other physiological processes as revealed by knock-out studies  
AUTHOR(S): Sadowski-Debbing, Kenneth; Coy, Johannes F.; Mier, Walter; Hug, Hubert; Los, Marek  
CORPORATE SOURCE: Clinic for Craniomaxillofacial Surgery, Ahaus, D-48683, Germany  
SOURCE: Archivum Immunologiae et Therapiae Experimentalis (2002), 50(1), 19-34  
CODEN: AITEAT; ISSN: 0004-069X  
PUBLISHER: Ossolineum Publishing House  
DOCUMENT TYPE: Journal; General Review  
LANGUAGE: English

AB A review with 113 refs. Caspases are crucial mediators of apoptosis, a form of physiol. cell **death**. Their activation is carefully controlled by a phylogenetically conserved **death** program, which is indispensable for the homeostasis and development of higher organisms. Dysregulation of apoptosis contributes to the pathogenesis of many human diseases. As effectors of the apoptotic machinery, caspases are considered potential therapeutic targets. In vitro studies have demonstrated the requirement of caspase activity for both the triggering phase as well as the execution of apoptosis, thus providing a mol. base for the fine-tuning of this process by pharmacol. agents. The precise roles of the individual caspases in vivo and their functional relation to each other have been best demonstrated in genetically modified animals. The generation of single caspase-deficient mice have confirmed most of the data obtained in vitro and exposed some new aspects previously undetected in the cell culture system. Interestingly, inactivation of many caspases revealed not only their expected participation in apoptotic events as well as in the maturation of **cytokines**, but also provided hints about the role of at least some caspases in cell differentiation and stimulatory responses. Here, the authors discuss what these studies have unveiled about the role of individual caspases in development, apoptosis, and inflammation, with particular focus on their role beyond the apoptotic process.